Amendments to the Specification:

Please amend the specification as shown:

1) Please delete the title of the invention found on page 1, lines 1-2 and replace it with the following title:

COMPOSITIONS FOR INHIBITING THE AGGREGATION PATHWAY OF α-SYNUCLEIN

2) Please delete the text under the "Brief Description Of The Drawings" found on page 5, lines 17 thru 26 and page 6, lines 1 thru 18,and replace it with the following:

Figure 1 consists of various diagrammatic representations illustrating α-synuclein increases iron dependent toxicity wherein Fig. 1A illustrates BE-M17 cells over-expressing wildtype, A53T or A30P α-synuclein treated with varying doses of $FeCl_2$ for 48 hrs and the viability was determined using the MMT assay; Fig. 1B illustrates BE-M17 cells over-expressing wildtype, A53T or A30P α-synuclein treated with varying doses of $FeCl_2$ for 48 hrs and the viability was determined using the LDH assay; Fig 1C illustrates BE-M17 cells over-expressing wildtype, A53T or A30P α-synuclein treated with varying doses of H_2O_2 for 48 hrs and the viability was determined using the MMT assay; Fig D illustrates in a, b, c and d the sequestration of iron due to α-synuclein.; *$p<0.01$, +, $p<0.01$ by ANOVA analysis.

Figure 2 consists of diagrammatic representations illustrating that iron binds to α-synuclein wherein Fig 2A $FeCl_2$ quenches the fluorescence emission spectrum by tyrosine in α-synuclein ($\lambda ex=280nm$); high doses of iron (>10mM) will quench tyrosine fluorescence, however proteins that bind iron exhibit quenching at much lower doses (presumably because the iron is kept near the tyrosine by the protein binding, tyrosine having a fluorescence emission spectrum that has a peak emission of 310 nm when excited at 280 nm, respectively; and wherein Fig 2B shows dose response curves for iron binding to wildtype and ΔC_{1-113} α-synuclein based on fluorescence emissions, wherein a

deletion construct lacking the last 27 amino acids of α-synuclein and analyzed binding of this construct, and a C-terminal construct of α-synuclein ΔC_{1-113} showed over a 4-fold reduction in iron binding, with an $IC_{50} = 726\mu M$ ($P<0.001$).

Figure 3 consisting of representative gels illustrating that magnesium protects against synuclein aggregation wherein Fig. 3A illustrates that magnesium converts α-synuclein to a conformation that resists aggregation and Fig 3B illustrating magnesium inhibits α-synuclein aggregation; magnesium (0.1mM) inhibits iron-induced aggregation of recombinant wild type α-synuclein in primary neurons where higher doses are needed because the cell membrane poses a barrier to passage of the ions and similar results are seen in BE-M17 cells over-expressing α-synuclein.

On page 26, line 18, after the word treatment, please insert the following: (see Fig. 1D).